

groups in terms of age, hyperlipidemia, body mass index, smoking and family history. Left ventricular global longitudinal strain and circumferential strain levels were significantly impaired in group 1 compared to group 2 (14.7 ± 3.3 , 22.6 ± 2.3 ; $p<0.001$ and 15.3 ± 2.7 , 20.7 ± 2.8 ; $p<0.001$, respectively) whereas no significant difference was observed between groups in terms of radial strain levels (36.5 ± 2.7 , 37.4 ± 3.3 ; $p=0.146$). Left ventricular global longitudinal and circumferential strain values were 14.2 ± 3.3 vs 15.1 ± 2.8 , respectively in the subgroup with high normal blood pressure and 15.3 ± 3.2 and 15.6 ± 2.7 , respectively in the subgroup with normal blood pressure, however there was no significant statistical difference ($p:0.162$ and $p:0.422$, respectively).

Conclusion: Left ventricular longitudinal and circumferential strain are impaired in patients with prehypertension, compared to patients with optimal blood pressure.

OP-035

Complexity of Hypertension is Perceived Through the Genetic Interactions of Multiple Genes

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Background: Hypertension being a multigenetic disease, the gene-gene interactions (epistasis) among the variants would consolidate global overview on gene-disease interactions.

Methods: Genetic variants in angiotensin converting enzyme (ACE), Angiotensin II receptor, type-1 (AT1R), angiotensinogen (AGT), aldosterone synthase (CYP11B2), endothelial nitric oxide synthase (NOS3), β_2 -adrenergic receptor (ADRB2), fat mass obesity associated gene (FTO) and guanine nucleotide binding protein, β -polypeptide 3 (GNB3) belonging to pathways like renin-angiotensinogen-aldosterone system (RAAS), kallikrein-kinin system (KKS), adrenergic receptor, nervous system and G protein coupled receptors family were screened using PCR-RFLP and SNaShot methods in age- and -ethnicity matched case-control 1500 north-Indian subjects. The study was approved by Human ethics committee.

Results: Gene-gene interactions revealed few best disease predicting models ($p<0.01$) such as the ACE I/D, AT1R 1166A/C and AGT -532C/T, -20A/C, -6G/A 235M/T; CYP11B2 -344T/C, Iw/Ic and NOS3 -922A/G, -786T/C, 4b/4a, 894G/T; FTO rs8050136C/A and GNB3 rs1129649T/C, 825C/T. The ORs (odds ratios) for CYP11B2 and NOS3 risk interacted-haplotypes (-344T/Ic) + (-922A/-786T/4a/894G) and (-344T/Ic) + (-922G/-786C/4a/894G) were 5.3 and 3.9, respectively ($p<0.04$); whereas the OR for protective interacted-haplotypes (-344T/Iw) + (-922A/-786T/4b/894G) was 0.7 ($p=0.03$). Furthermore, stratification of interacted 4- and 5-locus models comprising polymorphisms of ADRB2 and NOS3 on the basis of increasing number of risk alleles demonstrated remarkable augmentation in the OR from 1.3 to 14.2 ($p=0.508$ - $4.51E-07$) for hypertension susceptibility compared to respective individual gene models. Likewise stratification of 3-locus best model of FTO rs8050136C/A and GNB3 rs1129649T/C and 825C/T on the basis of interacted-genotypes comprising 1, 2, 3, 4 and 5 risk alleles correlated linearly with increased ORs from 1.9 to 11.6 for hypertension ($p<0.01$) compared to interacted-genotypes devoid of risk alleles. Of note, the genetic outcomes were further consolidated by significant varied clinical, biochemical parameters and expression levels, ($p<0.05$).

Debates: This study demonstrated the importance of non-linear interactions (epistasis) among the genes of several pathways in hypertension pathophysiology. A global cumulative consensus will immensely benefit the diagnosis and therapeutics.

OP-036

Estimated Glomerular Filtration Rate is Associated with Both Arterial Stiffness and N Terminal Pro-brain Natriuretic Peptide in Newly Diagnosed Hypertensive Patients

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Background: Even a slight decrease in the glomerular filtration rate (GFR) is an independent risk factor for cardiovascular disease. Arterial stiffness, left ventricular hypertrophy and N-terminal pro-brain natriuretic peptide (NT-proBNP) are independent risk factors for cardiovascular disease, which are particularly common in end-stage renal disease. We aimed to evaluate the association between GFR with arterial stiffness, left ventricle mass (LVM) and NT-proBNP in hypertensive subjects with normal to mildly impaired renal function.

Methods: The study population consisted of 285 newly diagnosed hypertensive patients (Mean age; 49.9 ± 11.8 years). GFR was estimated (eGFR) by the Modification of Diet in Renal Disease formula. Pulse wave velocity (PWV) and augmentation index (AIx) which reflects arterial stiffness, were calculated using the single-point method via the Mobil-O-Graph® ARCSolver algorithm. LVM was obtained by echocardiography. Plasma NT-proBNP was measured by electrochemiluminescence. The patients were divided into two groups according to the median eGFR value (eGFRlow group <101 and eGFRhigh group ≥ 101).

Results: LVM and NT-proBNP values were higher in eGFRlow group compared with eGFRhigh group ($p<0.05$). Pulse wave velocity and augmentation index values were higher in eGFRlow group compared with eGFRhigh group ($p<0.05$, for all) (Table). Multiple linear regression analysis showed that eGFR was independently associated with PWV ($\beta=-0.422$, $p<0.001$) and NT-proBNP ($\beta=-0.404$, $p<0.001$).

Conclusions: Present study showed that eGFR was independently associated with PWV and NT-proBNP values. Importantly, these findings may explain, in part, the increase in cardiovascular risk in with slightly impaired renal function.

Table. Comparison of baseline, laboratory, echocardiographic and arterial stiffness parameters between the groups

Variables	GFRhigh Group (n 143)	GFRlow Group (n 142)	P value
Age (Years)	43.1 \pm 9.0	56.9 \pm 10.1	<0.001
LVM (g)	168.0 \pm 44.6	183.4 \pm 46.1	0.004
NT-proBNP (pg/ml)	50.6 \pm 38.9	117.6 \pm 69.5	<0.001
PWV (m/s)	6.79 \pm 1.02	8.58 \pm 1.53	<0.001
AIx (%)	27.6 \pm 12.0	30.9 \pm 11.6	0.020

LVM; left ventricular mass, NT-proBNP; N terminal pro-brain natriuretic peptide, PWV; pulse wave velocity, AIx; augmentation index

OP-037

The Relationship among Thiazide Like Diuretic, Uric Acid and Erectile Dysfunction in Hypertensive Subjects

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Objective: Beta blockers and diuretics are frequently associated with erectile dysfunction (ED). However, the definitive mechanisms of ED secondary to these drugs are still unclear. Uric acid is a novel marker of atherosclerosis and endothelial dysfunction. Also, endothelial dysfunction plays a major role in ED. A common adverse effect of thiazide like diuretics is increase in serum uric acid level. We aimed to examine association between thiazide like diuretic uric acid and erectile dysfunction in hypertensive patients.

Method: A total of 200 current hypertensive patients (age between 30-70 years) were enrolled in this prospective study. ED was assessed by using SHIM test. After, demographic characteristics of patients were recorded; serum uric acid levels and the other laboratory analysis were performed following 12 hours fasting. Office blood pressure (BP) was measured and a detailed data about antihypertensive treatments were recorded. Patients divided into two groups based on the SHIM test. Patients with SHIM score <21 defined as ED and SHIM score ≥ 21 defined as normal erectile function. Difference between ED and normal erectile function group was compared and predictors of ED were analyzed. The effect of thiazide-like diuretics on ED and uric acid level was also tested.

Results: Prevalence of ED was found 55.0%. A total of 72 patients (36%) were using thiazide like diuretic. Office BP level was comparable between groups. Beta blocker users, smokers and diabetics were frequent in ED group. Age, uric acid levels (6.20 ± 1.56 vs 5.44 ± 1.32 , $p=0.01$), GFR and fasting glucose levels were significantly increased in ED group. Demographic characteristics and laboratory findings of patients with and without ED were presented in Table1. Multiple logistic regression model showed that age (odds: 1.08 (1.04-1.14), $p=0.001$), smoking (odds: 2.33 (1.04-5.20), $p=0.04$), and uric acid (odds: 1.76 (1.28-2.41), $p=0.04$) were independent predictors of ED. Beta blockers and thiazide like diuretics did not reach statistically significance in regression model. However, uric acid levels were increased in patients using thiazide like diuretic compared to non-using patients (6.30 ± 1.5 vs 5.5 ± 1.5 mg/dl, $p=0.05$, respectively). A weak negative correlation was observed between SHIM score and uric acid level ($r=-0.204$, $p=0.01$). For indicating ED in ROC analysis, a cut-off value for uric acid level of greater than 5.2 had 76.2% sensitivity, 43.7% specificity, 62.9 positive predictive value, and 59.4 negative predictive value for prediction of ED (Figure1.)

Conclusion: A large number of hypertensive patients suffer from ED regardless BP levels. Uric acid is an independent predictor of ED and thiazide like diuretics mediated increase in serum uric acid levels may contribute to ED via endothelial dysfunction.